compared to 1/14 LGI1-Ab+patients(p=0.0024) and 1/12 healthy controls.

Conclusion Neuropathic pain may be present in both LGI-Ab + and CASPR2-Ab+patients, and is immunotherapy responsive. Serum IgG from CASPR2-Ab+patients more frequently bound sensory neurons and dorsal root ganglia, suggesting pathophysiological differences which may underlie the more severe pain in these patients.

008 DISEASE REACTIVATION AFTER CESSATION OF DISEASE-MODIFYING THERAPY IN RELAPSING-REMITTING MULTIPLE SCLEROSIS

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Objectives To evaluate the rate of return of disease activity after cessation of multiple sclerosis (MS) disease-modifying therapy.

Methods This was a retrospective cohort study from two large observational MS registries: MSBase and OFSEP. Patients with relapsing-remitting MS who had ceased a disease-modifying therapy and were followed up for the subsequent 12-months were included in the analysis. The primary study outcome was annualised relapse rate in the 12 months after disease-modifying therapy discontinuation stratified by patients who did, and did not, commence a subsequent therapy. The secondary endpoint was the predictors of first relapse and disability accumulation after treatment discontinuation.

Results 18 029 eligible treatment discontinuation epochs were identified for seven therapies. Rates of relapse started to increase 2-months after natalizumab cessation. Commencement of a subsequent therapy within 2-4 months reduced the magnitude of disease reactivation. After discontinuation of fingolimod, rates of relapse increased overall, and stabilised faster in patients who started a new therapy within 1-2 months. Magnitude of disease reactivation for other therapies was low, but reduced further by commencement of another treatment 1-10 months after treatment discontinuation. Predictors of relapse were higher relapse rate in the year before cessation, female sex, younger age and higher EDSS. Commencement of a subsequent therapy reduced both the risk of relapse (HR 0.76, CI 0.72-0.81) and disability accumulation (0.73, 0.65-0.80).

Conclusion Understanding the rate of disease reactivation after discontinuing different MS immunotherapies will help guide optimal wash-out times for therapeutic agents during treatment sequencing.

009 PREDICTING INFECTION RISK IN MULTIPLE SCLEROSIS PATIENTS TREATED WITH OCRELIZUMAB: A RETROSPECTIVE COHORT STUDY

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Objective To examine factors determining risk of self-reported infections and antimicrobial use in patients receiving Ocrelizumab for MS.

Methods Retrospective, observational cohort study conducted in Ocrelizumab-treated patients at the Royal Melbourne Hospital. The association of clinical and laboratory factors with self-reported infection rate and antimicrobial use were estimated using univariate and multivariable logistic regression models.

Results 185 patients were included in the study, and 176 infections were reported in 89 patients (46.1%), and in 47 patients (25.3%) antimicrobial use was identified. In univariate analyses, a higher serum IgA was associated with reduced odds of infection (OR 0.44, 95% CI 0.25 - 0.76). In multivariable analyses, older age (OR 0.94, 95% CI 0.88 - 0.99), higher serum IgA (OR 0.37, 95% CI 0.17 - 0.80) and higher serum IgG (OR 0.81, 95% CI 0.67 - 0.99) were associated with reduced odds of infection. Older age (OR 0.85, 95% CI 0.75 - 0.96) and higher serum IgA (OR 0.23, 95% CI 0.07 - 0.79) were associated with reduced odds of antimicrobial use, whilst longer MS disease duration (OR 1.22, 95% CI 1.06 - 1.41) and higher EDSS (OR 1.99, 95% CI 1.02 - 3.86) were associated with increased odds of antimicrobial use.

Conclusions Higher serum IgA, IgG and older age were associated with reduced odds of infection. Our findings highlight non-uniformity of infection risk in Ocrelizumab-treated MS patients, and substantiate the need to monitor immunoglobulin levels pre-treatment and whilst on therapy.

010 REAL-WORLD EXPERIENCE WITH OCRELIZUMAB IN THE MSBASE REGISTRY – AUSTRALIAN RRMS COHORT

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Introduction Ocrelizumab (OCR) is a humanised anti-CD20^{+} monoclonal antibody for the treatment of Multiple Sclerosis.